

AD-A165 869

HUMAN VASCULAR FLUID RESPONSES TO COLD STRESS ARE NOT
ALTERED BY COLD ACC. (U) ARMY RESEARCH INST OF
ENVIRONMENTAL MEDICINE NATICK MA A J YOUNG ET AL.
MAR 86 USARIEH-M-19/86

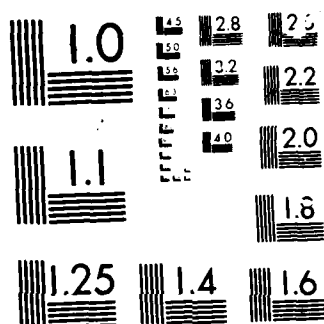
1/1

UNCLASSIFIED

F/G 6/19

NL





MICROCOPY RESOLUTION TEST CHART

AD-A165 869

DTIC FILE COPY

UNCLASSIFIED
SECURITY CLASSIFICATION OF THIS PAGE (When Data Entered)

REPORT DOCUMENTATION PAGE		READ INSTRUCTIONS BEFORE COMPLETING FORM
1. REPORT NUMBER M19/86	2. GOVT ACCESSION NO.	3. RECIPIENT'S CATALOG NUMBER
4. TITLE (and Subtitle) Human vascular fluid responses to cold stress are not altered by cold acclimation		5. TYPE OF REPORT & PERIOD COVERED
		6. PERFORMING ORG. REPORT NUMBER
7. AUTHOR(s) Andrew J. Young, Stephen R. Muza, Michael N. Sawka and Kent B. Pandolf		8. CONTRACT OR GRANT NUMBER(s)
9. PERFORMING ORGANIZATION NAME AND ADDRESS U.S. Army Research Institute of Environmental Medicine Natick, MA 01760-5007		10. PROGRAM ELEMENT, PROJECT, TASK AREA & WORK UNIT NUMBERS 3E162777A879 WU 127
11. CONTROLLING OFFICE NAME AND ADDRESS Same as 9. above		12. REPORT DATE March 1986
		13. NUMBER OF PAGES 22
14. MONITORING AGENCY NAME & ADDRESS (If different from Controlling Office)		15. SECURITY CLASS. (of this report)
		15a. DECLASSIFICATION/DOWNGRADING SCHEDULE
16. DISTRIBUTION STATEMENT (of this Report) Approved for public release; distribution is unlimited.		
17. DISTRIBUTION STATEMENT (of the abstract entered in Block 20, if different from Report)		
18. SUPPLEMENTARY NOTES		
19. KEY WORDS (Continue on reverse side if necessary and identify by block number) Hypothermia, plasma volume reduction, cold diuresis, body cooling		
20. ABSTRACT (Continue on reverse side if necessary and identify by block number) Repeated cold water immersion can induce the development of an insulative type of cold acclimation in man. This investigation determined if repeated cold water immersion produced changes in vascular fluid responses to cold stress in addition to the previously reported changes in thermoregulation. Seven male subjects performed a standareized cold air and cold water exposure before and again after a cold acclimation program. The cold acclimation program consisted of daily immersion (90 min) in cold water (18°C, stirred) repeated five times per week for five consecutive weeks. Cold acclimation did not alter the		

DTIC
ELECTE
MAR 27 1986
S E D

DD FORM 1473 EDITION OF 1 NOV 65 IS OBSOLETE

UNCLASSIFIED

SECURITY CLASSIFICATION OF THIS PAGE (When Data Entered)

86 3 26 049

UNCLASSIFIED

SECURITY CLASSIFICATION OF THIS PAGE(When Data Entered)

responses of plasma volume or electrolyte concentrations, nor urinary flow or electrolyte excretion during either cold air or cold water exposure. The percent reduction in plasma volume was larger ($P < 0.01$) in cold water (-17%) than in cold air (-12%). Cold water immersion resulted in greater ($P < 0.01$) diuresis than cold air exposure. Plasma K^+ concentration increased ($P < 0.01$) during cold (both air and water) exposure while plasma Na^+ concentration was unchanged. Calculated renal clearance and urinary excretion rate of both Na^+ and K^+ increased during cold (both air and water) exposure. The magnitude of plasma volume reduction during cold exposure was not correlated with either the degree of body cooling or diuresis. It is concluded that: (1) insulative cold acclimation does not influence vascular fluid responses to cold stress; and (2) although vascular fluid shifts, body cooling and diuresis are all greater in cold water than air, a consistent relationship among these parameters could not be established for an individual's response.

UNCLASSIFIED

SECURITY CLASSIFICATION OF THIS PAGE(When Data Entered)

HUMAN VASCULAR FLUID RESPONSES TO COLD STRESS
ARE NOT ALTERED BY COLD ACCLIMATION

Andrew J. Young, Stephen R. Muza,
Michael N. Sawka and Kent B. Pandolf

US Army Research Institute of Environmental Medicine
Natick, MA 01760-5007

Running Head: Vascular Fluid Responses to Cold

Address correspondence to:

Dr. Andrew J. Young
US Army Research Institute of
Environmental Medicine
Kansas St
Natick, MA 01760-5007
617-651-4837



Accession For	
NTIS GRA&I	<input checked="checked" type="checkbox"/>
DTIC TAB	<input type="checkbox"/>
Unannounced	<input type="checkbox"/>
Justification	
By	
Distribution/	
Availability Codes	
Dist	Avail and/or Special
A-1	

ABSTRACT

Repeated cold water immersion can induce the development of an insulative type of cold acclimation in man. This investigation determined if repeated cold water immersion produced changes in vascular fluid responses to cold stress in addition to the previously reported changes in thermoregulation. Seven male subjects performed a standardized cold air and cold water exposure before and again after a cold acclimation program. The cold acclimation program consisted of daily immersion (90 min) in cold water (18°C , stirred) repeated five times per week for five consecutive weeks. Cold acclimation did not alter the responses of plasma volume or electrolyte concentrations, nor urinary flow or electrolyte excretion during either cold air or cold water exposure. The percent reduction in plasma volume was larger ($P < 0.01$) in cold water (-17%) than in cold air (-12%). Cold water immersion resulted in greater ($P < 0.01$) diuresis than cold air exposure. Plasma K^{+} concentration increased ($P < 0.01$) during cold (both air and water) exposure while plasma Na^{+} concentration was unchanged. Calculated renal clearance and urinary excretion rate of both Na^{+} and K^{+} increased during cold (both air and water) exposure. The magnitude of plasma volume reduction during cold exposure was not correlated with either the degree of body cooling or diuresis. It is concluded that: (1) insulative cold acclimation does not influence vascular fluid responses to cold stress; and (2) although vascular fluid shifts, body cooling and diuresis are all greater in cold water than air, a consistent relationship among these parameters could not be established for an individual's response.

Key Words: Hypothermia, plasma volume reduction, cold diuresis, body cooling

INTRODUCTION

The effects of exposure to environmental extremes of heat and high altitude on human body fluid responses have been well studied; however, the effects of cold exposure on body fluid shifts have received less attention. Acute cold-air exposure has been generally reported to produce a reduction in plasma volume ranging from 7 to 15% (5,12). The techniques used to estimate plasma volume changes in these earlier studies are subject to quantitative error due to loss of intravascular protein or changes in erythrocyte volume. Nevertheless, the physiological responses to acute cold stress (e.g. shivering, vasoconstriction, diuresis) could all contribute to some degree of plasma volume reduction.

Previous investigations have not addressed the question as to whether, in awake man, the degree of body cooling is related to the magnitude of plasma volume reduction during cold exposure. Investigations employing animal models indicate that the plasma volume is progressively reduced as body temperature falls (7,9). However, it has also been observed that when body cooling is severe, fluid shifts may be reversed or absent (4,9). Hamlet (14) has suggested, although not experimentally substantiated, that body cooling is so rapid during immersion in cold water that vascular fluid shifts do not occur.

Although heat acclimation (23,24), altitude acclimation (26), and exercise training (8) all have been shown to influence vascular fluid regulation during exposure to environmental extremes, the influence of cold acclimation on body fluid responses to cold stress has not been addressed. The existence of true cold acclimation in man has been questioned, but strong evidence has recently been reported indicating that man can acclimate to cold (28). Repeated cold-water immersion was shown to produce altered physiological responses to cold-air exposure (e.g. lower skin temperatures and greater sympathetic nervous activity) indicative of the development of stronger cutaneous vasoconstriction

which improved tissue insulation (28). These alterations in physiological responses to cold exposure might also be associated with altered vascular fluid responses to cold stress. There is some evidence that ethnic or seasonal differences observed in blood volume may be related to chronic cold exposure (6). On the other hand, seasonal changes in blood volume may actually be due to changes in physical activity (13,27), length of daylight (25), or the induction or decay of heat acclimation (5), rather than cold acclimation. Other studies which have attempted to investigate changes in vascular fluid volumes during chronic cold exposure have employed studies of too short duration (3-5 days) to permit development of a significant degree of acclimation (16) or have been confounded by the effects of progressive hypohydration and caloric deficit (21).

The purpose of the present investigation was to examine, under controlled laboratory conditions, the effects of acute cold exposure and cold acclimation on human vascular fluid regulation. To determine the influence of cold stress intensity and body cooling rate on the magnitude of vascular fluid shifts, responses in cold air were compared to responses in cold water. In addition, the effects of an acclimation program consisting of repeated (intermittent) cold water immersion on vascular fluid responses to cold stress were studied.

METHODS

Subjects and Experimental Design. Seven male soldiers volunteered as subjects for this study after they had been completely informed about the requirements and risks of participation. Descriptive characteristics (mean \pm SE) of the subjects were: age = 24 ± 2 yrs; body mass = 70 ± 4 kg; body surface area = 1.98 ± 0.07 m²; body fat (hydrostatic weighing (11)) = $17.4 \pm 1.8\%$; mean skinfold thickness (14 sites) = 11.4 ± 1.5 mm; maximum aerobic power (treadmill

running (18)) = $45.3 \pm 1.6 \text{ ml}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$. The subjects were all caucasians and native to the continental United States. The subjects had participated in no significant cold weather activities for at least nine months prior to this study which was completed in Natick, MA during the late fall when seasonal effects of cold exposure were expected to be minimal.

The subjects completed a standardized cold-air exposure two days before and again two days after completion of a program of repeated cold-water immersions. Physiological responses measured during the first and last cold-water immersion were used for comparison with responses during the cold-air exposures. The standardized cold-air exposures consisted of a 30 min baseline period spent reclining in a nylon-mesh lounge chair in a comfortable environment ($T_a=24^\circ\text{C}$, $\text{rh}=30\%$) while wrapped in blankets. During the last five min of this period, a venous blood sample was obtained from an indwelling catheter previously placed in an antecubital vein. After the blood sample had been obtained, the subject stood, voided his bladder, entered the environmental chamber ($T_a=5^\circ\text{C}$, $\text{rh}=30\%$); and then reclined for 90 min wearing only swim trunks. Venous blood samples were obtained again during the last five min of the cold-air exposure. At the completion of the cold exposure period, the subjects again voided their bladder. The two cold (18°C , stirred) water exposures used for comparative purposes were performed using the same protocol as the cold-air exposure with the exception that the environmental conditions for the baseline period were slightly warmer ($T_a=27^\circ\text{C}$, $\text{rh}=65\%$). Rectal (T_{re}) and mean weighted skin (T_{sk}) temperatures were measured during the last three min of the baseline period and at two min intervals during the cold exposure period for both cold air-and cold-water tests.

The repeated cold-water immersion program consisted of a daily 90-min immersion in cold (18°C, stirred) water, repeated five times a week for five consecutive weeks. Generally, the immersion sessions were accomplished on five consecutive days each week (Mon-Fri). On occasion, individuals missed a scheduled immersion session and in that event, the missed session was completed on the weekend. Once during the study, a midweek holiday resulted in cancellation of a scheduled immersion which could not be rescheduled. Thus, the subjects completed a total of twenty-four cold-water immersions. While immersed, the subject reclined quietly on a nylon mesh lounge chair, and water level was adjusted to the base of the neck. Immersions were terminated after 90-min or if the subject's T_{re} fell below 35°C.

Experimental procedures. The subjects reported to the laboratory to be instrumented 1 hr prior to the experimental sessions. During this period, they consumed 500 ml of tap water. A thermister inserted 10 cm beyond the anal sphincter was used to measure T_{re} . During the cold air exposure, skin temperature was measured at three sites using thermocouples attached to the skin (forearm, chest, calf). A venous catheter was emplaced for blood sampling and kept patent with heparized saline. Immediately before the baseline period began, the subject voided his bladder and a stopwatch was started in order to measure the rate of urine formation during the baseline and subsequent cold exposure period. Aliquots of the total urine produced during each of the two periods were analyzed as described below.

Venous blood was analyzed in triplicate for hematocrit and in duplicate for hemoglobin concentration (Coulter Hemoglobinometer), plasma osmolality (Precision Systems Osmete A), plasma protein concentration (American Optical refractometer), and plasma Na^+ and K^+ concentration (NOVA Na^+/K^+ analyzer).

Aliquots of urine were analyzed in duplicate for Na^+ and K^+ concentration and specific gravity.

The percent changes in plasma volume and erythrocyte cell volume were calculated from the hematocrit and hemoglobin values obtained during the baseline and cold exposure period (10). Absolute plasma volume during the baseline period of the first cold air exposure was estimated from the equation of Allen et al. (2), and plasma volume for the remaining conditions was calculated by adjusting these initial values by the appropriate percent change in plasma volume. Urine flow rate for the baseline and the cold stress period was calculated by dividing the total urine collected during the period by the actual time elapsed since the previous voiding of the bladder. Urinary electrolyte clearance was calculated as the product of urine flow rate and the ratio between urinary electrolyte concentration and plasma electrolyte concentration.

Statistical analyses. Multifactor, repeated measures analysis of variance (ANOVA) was used to determine if the factors "exposure" (baseline versus cold exposure), "environment" (cold air versus cold water), or "acclimation" (pre-versus post-) had significant effects. In the event that ANOVA revealed significant main effects or multifactor interactions, Tukeys critical difference was calculated and used to locate significant differences between means. Complete measurements of urinary parameters were not obtained from one of the subjects, so urinary data of the remaining six was analyzed and reported. Single and multiple factor regression analysis was performed to compare the changes in plasma volume with changes in body temperature and the amount of urine formed during cold exposure. Unless otherwise noted, results are presented as the mean \pm SE. Thermoregulatory data have been presented and discussed in detail elsewhere (28).

RESULTS

The percent changes in plasma volume during each cold exposure relative to the basal condition are shown in Figure 1. Plasma volume decreased during both cold-air and cold-water exposure, but there was no significant acclimation effect. The percent decrease in plasma volume during cold-water immersion ($16.6 \pm 1.6\%$) was greater than ($P < 0.01$) that during cold-air exposure ($11.8 \pm 1.4\%$). The absolute plasma volumes calculated for the basal period prior to each cold stress were $2.94 \pm 0.12\text{l}$ (cold air) and $2.88 \pm 0.11\text{l}$ (cold water) for pre-acclimation and $2.92 \pm 0.14\text{l}$ (cold air) and $3.04 \pm 0.11\text{l}$ (cold water) for post-acclimation. These values of basal plasma volume were not significantly different. The changes in T_{re} during each cold stress (basal T_{re} - final T_{re}) were $+ 0.03 \pm 0.06$ °C (cold air) and -0.79 ± 0.15 °C (cold water) for preacclimation and -0.28 ± 0.14 °C (cold air) and -1.00 ± 0.27 °C (cold water) for postacclimation tests. Linear regression and analysis of covariance both failed to reveal any significant relationship between changes in plasma volume (% or absolute) with the change in T_{re} during exposure to either cold air or cold water.

Table 1 about here

In Table 1 are shown mean corpuscular hemoglobin, plasma protein, and electrolyte concentrations, plasma osmolarity and the calculated total circulating protein. Also shown in Table 1 are the results of the analysis of variance performed on the data. Acclimation had no significant effect on any of these vascular fluid components. Plasma protein concentration increased during cold exposure and there was a greater increment in plasma protein concentration during cold-water immersion than during cold-air exposure. Total circulating

plasma protein was not significantly altered during cold-air or cold-water exposure. Similarly, mean corpuscular hemoglobin concentration and plasma osmolarity were not affected by cold exposure (either air or water). While plasma Na^+ concentration was constant during both cold air and water exposure, plasma K^+ increased ($P < 0.01$) during cold stress (both air and water).

Table 2 about here

The effect of cold stress on urinary composition and flow rate is shown in Table 2. Again, cold acclimation had no effect on either urinary flow rate or composition during basal or exposure conditions. There were no significant differences between the basal values of any of the parameters shown in Table 2. Urine flow rate increased ($P < 0.01$) during cold exposure. With cold-air exposure, urine flow rate increased by 51% above basal flow, but a greater ($P < 0.01$) diuresis was occurred during cold-water immersion when urine flow rate increased by 388% above basal level. There was no significant relationship between the reduction in plasma volume (percent or absolute) and the magnitude of diuresis during cold exposure. Changes in urine specific gravity with the cold exposure followed the same pattern as did the changes in urine flow rate. Urine specific gravity fell during exposure to both cold air and cold water, but a greater ($P < 0.01$) fall in urinary specific gravity occurred during cold-water immersion than with cold-air exposure. The rate of excretion of both Na^+ and K^+ increased significantly with cold-air exposure. The increment in K^+ excretion rate was the same during cold-air exposure as during cold-water immersion; but the increment in Na^+ excretion was greater ($P < 0.01$) during cold water immersion than during cold-air exposure.

Table 3 about here

Table 3 shows renal electrolyte clearance for all the experimental conditions. Renal electrolyte clearance was not significantly affected by cold acclimation. During both cold-air exposure and cold-water immersion, plasma Na^+ and K^+ clearance increased ($P < 0.01$) significantly with no difference in the increment between water and air.

DISCUSSION

The thermoregulatory adaptations which occurred as a result of the repeated cold-water immersion program are discussed in detail in a separate report (28). Briefly, repeated cold water immersion resulted in development of an insulative type of cold acclimation similar to adaptations exhibited by Korean breath-hold divers before the use of wetsuits became common practice (19). This insulative cold acclimation is primarily characterized by dramatically lower steady state skin temperature during cold stress following the cold-water immersion program. The lower skin temperature during postacclimation cold exposure was attributed to a greater cutaneous vasoconstriction mediated by more pronounced sympathetic nervous stimulation. The latter was evidenced by the observation that there was a larger increment in circulating norepinephrine during cold exposure after acclimation.

The effect of induced cold acclimation on body fluid regulation in humans under basal conditions or subsequent to cold exposure has not been previously reported. The results of the present investigation indicate that insulative cold acclimation produced by intermittent, and (relatively) short exposures to severe

cold stress had no measurable effect on body fluid regulation. In this respect, acclimation induced by cold-water immersion is similar to the effects of long term (11 days to longer) exposure to cold which is generally reported to have no effect on body fluid regulation (12). Short or intermediate (24 hours to 7 days) exposure to continuous cold has been reported to result in a reduction in plasma and blood volume in man (12). However, in those studies that employed short or intermediate cold exposure, the subjects maintained a negative water balance; thus the alteration in plasma and blood volume observed during short term cold exposure is probably not a direct effect of cold, but rather likely to be an effect of hypohydration. Hypohydration will reduce resting plasma volume as well as alter the vascular fluid shifts which occur with acute heat stress (22).

One study (20) has been reported in which plasma volume responses to cold stress were found to be different in acclimatized compared to nonacclimatized subjects, and differences in hydration should not have been a confounding factor. Rochelle and Horvath (20) reported that surfers, who had been chronically exposed to cold water, experienced a smaller plasma volume reduction during cold stress as compared to control subjects. However, those investigators reported that there was a significant correlation between the percent decrease in plasma volume and the percent body fat. When the data reported by Rochelle and Horvath (Table 1 and 2, Ref 20) are reanalyzed using analysis of covariance to remove the effect of body fat, the difference in plasma volume reduction between acclimatized and nonacclimatized subjects is not statistically significant. In the present study, body fat assessment was performed only once; however, total body mass was measured weekly and before each of the four standardized cold exposures. Since there were no significant changes in total body mass, it is likely that the subjects' body composition was constant

throughout the study. In sum, it seems that cold acclimation/acclimatization per se does not influence vascular fluid shifts as long as hydration level and body composition remain stable during the acclimation period.

Acute exposure to both cold air and cold water resulted in a plasma volume reduction while total circulating protein remained constant. Cold-induced plasma volume reduction has been previously reported (5,12,20). It was thought that comparison of the same subjects' vascular fluid responses in two different cold stress mediums might provide information concerning the factors which determine the magnitude of plasma volume reduction. Heat loss during cold-water immersion is more severe than cold-air exposure due to the greater conductive heat transfer properties of water. Thus the degree of cold stress, as reflected by the reduction in T_{re} , might be related to the magnitude of the concomitant plasma volume reduction during cold exposure. A greater drop in T_{re} was seen in cold water ($\sim 1.0^{\circ}\text{C}$) than in cold air ($> 0.3^{\circ}\text{C}$), and the plasma volume reduction was also greater during cold-water immersion compared to cold air (see Figure 1). However, analysis of covariance and linear regression indicated no significant correlation between the decrease in an individual's T_{re} and the corresponding plasma volume reduction during cold exposure.

Three physiological responses to cold stress that have been postulated to be related to the reduction in plasma volume are: shivering (9), diuresis (1) and sequestration of vascular fluid in certain regions of the vascular system (7). Shivering, like other forms of muscular exercise, could result in intracellular accumulation of metabolic catabolites (e.g. lactic acid) which increase intracellular anion concentration or osmolality, thereby drawing fluid from the plasma into the cells (17). The results of the present investigation indicate that shivering alone does not account for the plasma volume reduction during cold

stress. The O_2 uptake during cold exposure can be used to quantitate shivering (see ref 23 for data and measurement details), and no significant correlation was found between an individual's O_2 uptake and the plasma volume reduction during cold exposure. Furthermore, since both the plasma K^+ concentration and urinary K^+ excretion increased during the cold exposures, water is apparently not entering the cells; rather intracellular fluid volumes probably decreased during cold exposure. Intuitively, it seems obvious that diuresis during cold exposure should contribute to and be at least somewhat related to the plasma volume reduction, especially since there was a greater diuresis during cold-water immersion than during cold-air exposure. However, linear regression again failed to indicate a significant correlation between the volume of an individual's urine output and the plasma volume reduction during cold exposure. This finding is consistent with the observation that plasma volume is reduced during cold exposure even when diuresis is inhibited by administration of vasopressin (3). Finally, an attempt was made to account for the combined effects of diuresis and shivering on the plasma volume reduction during cold exposure using multiple regression analysis, and no significant combined effect was indicated. Failure to demonstrate statistically significant relationships may be due to the relatively small sample size or the sensitivity of measurements.

Another consideration is that the calculation of the percent change in plasma volume using changes in the venous hematocrit and hemoglobin concentration may not be valid under cold exposure conditions. This method of calculating changes in plasma volume requires the assumption that F-cell ratio remains constant (10). The F-cell ratio is the ratio between overall hematocrit and venous (large vessel) hematocrit, and provides an index of erythrocyte distribution between large vessels (higher hematocrit) and small vessels (lower

hematocrit). The intense vasoconstriction produced by cold stress will reduce the vascular volume of small vessels being perfused thereby lowering (relative to normothermic) the venous hematocrit measured at a given overall hematocrit (increased F-cell ratio). Thus, if F-cell ratio does increase during cold exposure, the use of changes in venous hematocrit and hemoglobin values to calculate plasma volume changes may result in underestimation of the magnitude of actual hemoconcentration: (a) from normothermic to hypothermic conditions, (b) between cold-air and cold-water exposure, and (c) from pre- to postacclimation. Research is needed to determine the effect of cold exposure on F-cell ratio of awake humans.

Little attention has been focused on the effects of cold exposure on plasma and urinary electrolytes in man. Both cold-air exposure and cold-water immersion produced an increase in plasma K^+ concentration, but plasma Na^+ concentration remained unchanged. These findings are in contrast to those of Lennquist et al. (16) who reported that cold-air exposure ($15^{\circ}C$) had no effect on plasma K^+ and produced a decrease in plasma Na^+ . However, their subjects were exposed to continuous cold for periods ranging from 1 to 6 days and plasma electrolytes were measured only at 24 hour intervals; the decrease in plasma Na^+ did not become apparent until about the third day (16). An increase in plasma K^+ concentration has been reported to occur in man under conditions of profound hypothermia (induced for surgical purposes) but not until core temperatures declined to about $25^{\circ}C$ (10). The increase in plasma K^+ observed during cold exposure in the present study was not due to increased renal reabsorption since both urinary excretion (Table 2) and calculated renal clearance (Table 3) of K^+ increased during cold exposure. It seems likely that the increase in plasma K^+ results from a reduction in the cellular Na^+/K^+ ATPase activity of cooled

tissues. Reduced Na^+/K^+ ATPase activity would allow K^+ leaking from cells to accumulate in the plasma. It is possible that the increase in plasma K^+ with acute cold exposure is a transient response. An increase in plasma K^+ would stimulate aldosterone release which in turn would increase Na^+/K^+ ATPase activity thereby eventually reestablishing normal electrolyte balance.

In summary, acute cold exposure (either air or water) results in a reduction in plasma volume concomitant with increased urine output. The hemoconcentration is isosmotic and not accompanied with loss of circulating plasma protein. Cold acclimation does not influence the cold induced plasma volume changes or diuresis. Despite the fact that changes in body temperature, plasma volume and urine output are greater in cold water than in cold air, no direct relationship between these parameters could be established.

Acknowledgements

The authors thank the volunteers for their outstanding performance. The expert technical assistance of James Bogart, Bruce Cadarette, Leslie Levine, Nancy Pimental and Karen Speckman, the statistical services of Tammy Doherty, and preparation of the manuscript by Dorothy Leader are all gratefully acknowledged.

The opinions and/or findings presented in this paper are these of the authors and should not be construed as Official Department of the Army position, policy or decision. Human subjects participated in these experiments after giving their free and informed consent. Investigators adhered to AR 70-25 and USAMRDC Regulation 70-25 on Use of Volunteers in Research.

Publication approved; distribution unlimited.

References

1. Adolph, E.F. and G.W. Molnar. Exchanges of heat and tolerances to cold in men exposed to outdoor temperature. *Am. J. Physiol.* 146:507-537, 1946.
2. Allen, T.H., M.T. Peng, K.P. Chen, T.C. Huang, C. Chang and H.S. Fang. Prediction of blood volume and adiposity from body weights and cube of height. *Metabolism.* 36:128-144, 1956.
3. Bader, R.A., J.W. Eliot and D.E. Bass. Hormonal and renal mechanisms of cold diuresis. *J. Appl. Physiol.* 4:649-658, 1952.
4. Barbour, H.G., E.A. McKay and W.F. Griffith. Water shifts in deep hypothermia. *Am. J. Physiol.* 140:9-19, 1943.
5. Bass, D.E., and A. Henschel. Responses of body fluid compartments to heat and cold. *Physiol. Rev.* 36:128-144, 1956.
6. Bazett, H.C., F.W. Lunderman, J. Doupe and J.C. Scott. Climatic effects on the volume and composition of blood in man. *Am. J. Physiol.* 129:69-83, 1940.
7. Chen, R.Y.Z. and S. Chien. Plasma volume, red cell volume, and thoracic duct lymph flow in hypothermia. *Am. J. Physiol.* 233:4605-4612, 1977.
8. Convertino, V.A., L.C. Keil and J.E. Greenleaf. Plasma volume, renin and vasopressin responses to graded exercise after training. *J. Appl. Physiol.* 54:508-514, 1983.
9. D'Amato, H.E. Thiocyanate space and distribution of water in the musculature of the hypothermic dog. *Am. J. Physiol.* 178:143-147, 1954.
10. Dill, D.B. and D.L. Costill. Calculation of percentage changes in volumes of red blood cells and plasma in dehydration. *J. Appl. Physiol.* 37:247-248, 1974.

11. Durnin, J.V.G.A. and J. Wommersley. Body fat measured from total body density and its estimate from skinfold thickness on 481 men and women aged 16 to 72 years. *Br. J. Nutr.* 32:77-97, 1974.
12. Fregley, M.J. Water and electrolyte balance during exposure to cold. *Pharmac. Ther.* 18:199-231, 1982.
13. Greenleaf, J.E. Physiological responses to prolonged bed rest and fluid immersions in humans. *J. Appl. Physiol.* 57:619-633, 1984.
14. Hamlet, M.P. Fluid shifts in hypothermia. In: *The Nature and Treatment of Hypothermia*. R.S. Pozoo and L.E. Witness (eds). Univ Mn Press, Minneapolis, MN. 94-97, 1978.
15. Langdon, L. and D.P.E. Kingsley. Changes in serum and urinary potassium levels during profound hypothermia in man. *J. Clin. Path.* 17:257-259, 1964.
16. Lennquist, S., P.O. Granberg and B. Weilin. Fluid balance and physical work capacity in humans exposed to cold. *Arch. Environ. Health.* 29:241-249, 1974.
17. Lundvall, J., S. Mellander, H. Westling and T. White. Fluid transfer between blood and tissues during exercise. *Acta Physiol. Scand.* 85:258-269, 1972.
18. McArdle, W.O., F.I. Katch and G.S. Peacher. Comparison of continuous and discontinuous treadmill and bicycle tests for max $\dot{V}O_2$. *Med. Sci. Sports.* 5:156-160, 1973.
19. Park, Y.S., D.W. Rennie, I.S. Lee, Y.D. Park, K.S. Park, D.H. Kang, D.J. Suh, S.H. Lee, S.Y. Hong and S.K. Hong. Time course of deacclimatization to cold water immersion in Korean women divers. *J. Appl. Physiol.* 54:1708-1716, 1983.

20. Rochelle, R.D. and S.M. Horvath. Thermoregulation in surfers and non-surfers immersed in cold water. *Undersea Biomed. Res.* 5:377-390, 1978.
21. Rogers, T.A., J.A. Satliff and J.C. Klopping. Energy cost, fluid electrolyte balance in subarctic survival situations. *J. Appl. Physiol.* 19:1-8, 1984.
22. Sawka, M.N., R.P. Francesconi, N.A. Pimental and K.B. Pandolf. Hypohydration and vascular fluid shifts during exercise in the heat. *J. Appl. Physiol.* 56:91-96, 1984.
23. Senay, L.C. Plasma volume and constituents of heat exposed men before and after acclimatization. *J. Appl. Physiol.* 57:570-575, 1975.
24. Senay, L.C., D. Mitchell and C.H. Wyndham. Acclimatization in hot humid environments: body fluid adjustments. *J. Appl. Physiol.* 40:786-796, 1976.
25. Sjostrand, T. Volume and distribution of blood and their significance in regulating circulation. *Physiol. Rev.* 33:202-228, 1953.
26. Surks, M.I., K.S.K. Chinn and L.O. Matoush. Alterations in body composition in man after acute exposure to high altitude. *J. Appl. Physiol.* 21:1741-1746, 1966.
27. Wilson, O. Physiological changes in the blood in the Antarctic: A Preliminary Report. *Br. Med. J.* 2:1425-1428, 1953.
28. Young, A.J., S.R. Muza, M.N. Sawka, R.R. Gonzalez and K.P. Pandolf. Human thermoregulatory responses to cold air are altered by repeated cold water immersion. *J. Appl. Physiol.* (in press), 1986.

Table 1. Effects of cold exposures on vascular fluids and components.

Environment	PREACCLIMATION		POSTACCLIMATION		FACTOR MAIN EFFECTS			FACTOR Inter-Actions
	Basal	Exposure	Basal	Exposure	Environment (A)	Acclimation (B)	Exposure (C)	
Plasma Protein, g·100ml ⁻¹								
Cold Air	7.18±0.17	8.02±0.14	7.07±0.12	8.02±0.09	NS	NS	Exposure > Basal*	AxC†
Cold Water	7.04±0.10	8.26±0.11	6.89±0.11	8.05±0.13				
Total Plasma Protein, gm								
Cold Air	210.2±6.2	209.8±7.1	205.7±8.8	209.9±9.1	NS	NS	NS	NS
Cold Water	202.4±5.5	199.7±5.2	208.3±7.3	209.9±7.2				
Plasma Osmolality								
Cold Air	287.2±2.0	286.2±1.7	286.2±1.2	288.1±1.2	NS	NS	NS	NS
Cold Water	285.6±1.1	290.1±1.8	291.6±1.5	283.1±11.0				
Mean Corpuscular Hb, g·100ml ⁻¹								
Cold Air	34.2±0.4	34.5±0.3	36.8±0.3	36.8±0.4	NS	NS	NS	NS
Cold Water	36.4±0.3	35.8±0.3	36.9±0.2	36.3±0.3				
Plasma Na ⁺ , meq·ℓ ⁻¹								
Cold Air	144.2±1.0	144.4±0.6	144.6±0.4	144.9±0.3	NS	NS	NS	NS
Cold Water	144.2±0.3	145.1±0.4	144.6±0.5	145.7±0.6				
Plasma K ⁺ , meq·ℓ ⁻¹								
Cold Air	4.1±1.0	4.6±0.1	4.3±0.1	4.8±0.1	NS	NS	Exposure > Basal*	NS
Cold Water	4.1±0.1	4.7±0.2	4.1±0.1	4.6±0.1				

Values are mean ±SE (N=7). *Significant at P ≤ 0.01; †Significant at P ≤ 0.05.

Table 2. Effects of cold exposure on urinary fluid and electrolytes.

Environment	PREACCLIMATION		POSTACCLIMATION		FACTOR MAIN EFFECTS			FACTOR Inter-Actions
	Basal	Exposure	Basal	Exposure	Environment (A)	Acclimation (B)	Exposure (C)	
Urine flow rate, ml·min ⁻¹								
Cold Air	3.2±0.9	4.4±0.6	2.5±0.7	4.2±0.8	NS	NS	Exposure > Basal*	AxC*
Cold Water	1.1±0.4	4.7±0.5	1.3±0.2	7.0±1.1				
Urine Specific Gravity								
Cold Air	1.012±0.003	1.008±0.002	1.015±0.003	1.010±0.002	NS	NS	Exposure < Basal	AxC*
Cold Water	1.020±0.003	1.006±0.001	1.021±0.002	1.008±0.002				
Na ⁺ Excretion Rate, meq·min ⁻¹								
Cold Air	0.18±0.05	0.30±0.04	0.21±0.04	0.36±0.05	NS	NS	Exposure > Basal*	AxC*
Cold Water	0.09±0.03	0.28±0.04	0.17±0.02	0.53±0.07				
K ⁺ Excretion Rate, meq·min ⁻¹								
Cold Air	0.09±0.02	0.15±0.02	0.07±0.01	0.15±0.01	NS	NS	Exposure > Basal*	NS
Cold Water	0.04±0.01	0.13±0.02	0.04±0.01	0.15±0.01				

Values are means ±SE (N=6). *Significant at P ≤ 0.01.

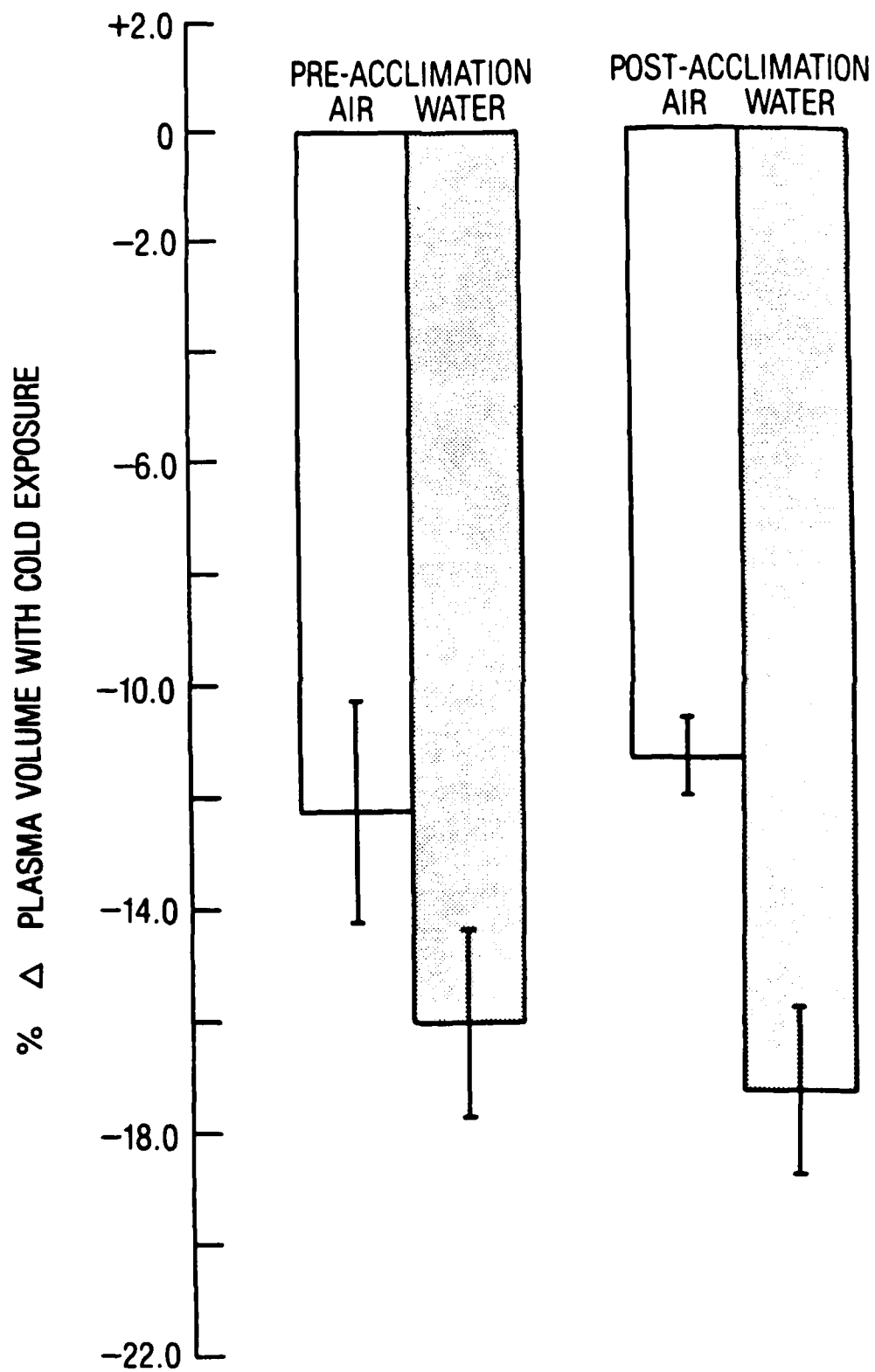
Table 3. Effects of cold exposure on renal electrolyte clearance.

Environment	PREACCLIMATION		POSTACCLIMATION		FACTOR MEAN EFFECTS			FACTOR Inter-Actions
	Basal	Exposure	Basal	Exposure	Environment (A)	Acclimation (B)	Exposure (C)	
Na ⁺ clearance, ml·min ⁻¹								
Cold Air	1.29±0.38	2.07±0.85	1.43±0.28	2.56±0.35	NS	NS	Exposure > Basal*	NS
Cold Water	0.65±0.20	1.94±0.30	1.13±0.17	2.75±0.25				
K ⁺ clearance, ml·min ⁻¹								
Cold Air	20.02±4.72	31.85±3.72	16.20±2.58	29.77±2.08	NS	NS	Exposure > Basal*	NS
Cold Water	9.75±3.41	26.27±4.76	9.15±1.57	29.16±4.87				

Values are mean ±SE (n=6), *Significant at P < 0.01.

FIGURE LEGEND

FIGURE 1. Percent changes in plasma volume during cold air and cold water exposure. Values are mean \pm SE (N=7).



END
FILMED

4-86

DTIC